# Radiofrequency Catheter Cure of Idiopathic Ventricular Tachycardia — First Experience in Hawaii

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*Radiofrequency catheter ablation is a well-established technique in the therapy of many forms of supraventricular tachyarrhythmias. Its usage in ventricular arrhythmias is little known. This is a report of its first usage in Hawaii in the treatment of 2 specific forms of ventricular tachycardia in 5 patients without structural heart disease. A cure was achieved in 4 of the 5 patients.*

## Introduction

Therapy for ventricular tachycardia (VT) has traditionally been problematic. Drugs have low efficacy and a high side effect profile, including life-threatening proarrhythmia.[1-2] Device therapy is effective, but is at best suppressive with considerable cost and morbidity, and the long-term effectiveness still awaits better definition with randomized, controlled trials.[3-4] Surgery eliminates the arrhythmia circuit in selected patients, but incurs high cost, morbidity, and acute mortality (mean 12%).[5] Catheter ablation, pioneered by Professor Melvin Scheinman from University of California at San Francisco, is a major step forward in the treatment of arrhythmias,[6] promising cure with abbreviated hospitalization and lower cost. This technique initially employed direct current (DC) energy, and its usage was confined to supraventricular tachyarrhythmias. Hartzler, the renowned coronary angioplasty expert, was the first to report DC catheter ablation for VT.[7]

Unfortunately, this approach is plagued by several serious problems, including cardiac perforation and cardiogenic shock. In large part, these complications are due to the nature of the energy source—DC discharge is relatively *uncontrolled*, and the release of an electrical charge in blood causes electrolysis and gas formation, which in turn can lead to *barotrauma.* In comparison, catheter ablation with radiofrequency (RF) is more controlled and gives rise to discrete lesions with well-demarcated borders, without gas formation or destruction of the blood elements. Its usage in the past several years has already revolutionalized the therapy for supraventricular tachycardias (SVT).[8-10] The experience with this form of energy in VT ablation still is very limited.

The following report of a consecutive series of 5 patients demonstrates the usefulness of the technique in the elimination of 2 unusual forms of VT, both occuring in patients without overt structural heart disease. These 2 arrhythmias arise from the right ventricular outflow (RVOT) or from the basal left ventricle (LV), at the region of the posterior fascicle.

## Materials and Methods

To be eligible for the procedure, a patient must have significantly symptomatic arrhythmia which is also documented by ECG or Holter monitoring. To rule out organic heart disease as a cause for the VT, the patient would need to have a normal treadmill test and echocardiogram, or a normal cardiac catheterization. If the VT were of RV origin, an MRI scan would be performed to rule out arrhythmogenic RV dysplasia.

After informed consent is obtained, the patient would undergo a baseline electrophysiologic study. In most cases (4/5 patients), the ablative attempt was performed in the same procedure as the baseline study. Quadripolar electrode catheters were introduced percutaneously through sheaths inserted into the left or right femoral veins by the Seldinger technique. They were variably positioned across the tricuspid valve for His bundle recordings, in the high right atrium, and in the right ventricle, generally in the apex. On one occasion, a catheter was advanced into the coronary sinus to outline the mitral annulus. In the baseline study, VT induction was performed by RV overdrive pacing or by programmed stimulation using a basic drive train of 8 beats (at cycle lengths of 500 or 400 milliseconds) and with up to 3 ventricular extrastimuli. If programmed electrical stimulation (PES) was unsuccessful, an isoproterenol infusion would be given at 1 mcg to 4 mcg per minute, and PES repeated as necessary.

After the tachycardia was induced, a 12 lead ECG was obtained to define the tachycardia morphology (Fig 1). The ventricular nature of the tachycardia was demonstrated by either ventriculo-atrial dissociation or retrograde Hisian depolarization. Then endocardial activation mapping would be performed. In the case of the fascicular tachycardia, another quadripolar catheter would be introduced into the left ventricle through the

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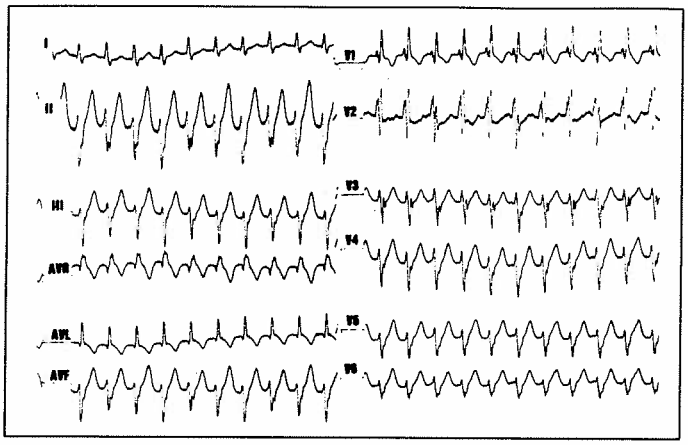

Fig 1.—Induced ventricular tachycardia from Case 1 (identical to clinical tachycardia) arising from basal inferior left ventricle. Note right bundle block configuration with superior axis.

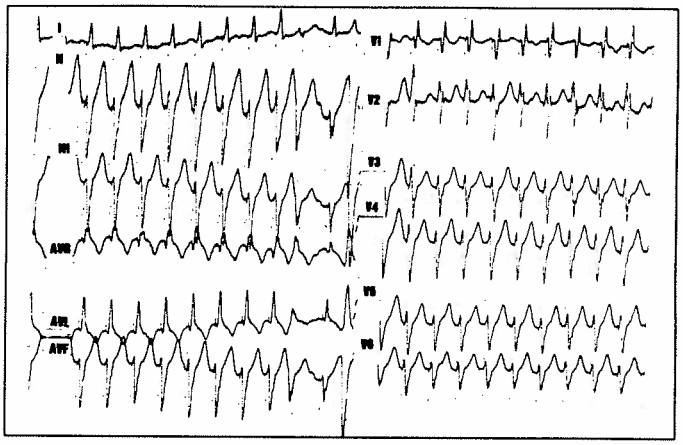

Fig 2.— Pace map produced by pacing from the tip of the ablation catheter in Case 1. Note 12 out of 12 ECG lead concordance with the tachycardia recorded in Fig 1.

aortic valve, via femoral arterial access. After the origin of the VT was approximately determined, the mapping catheter was substituted with the ablation catheter, which was also a quadripolar electrode catheter but with an enlarged deflectable 4 mm or 5 mm tip (Mansfield or EPT). This was advanced to the site of presumed VT focus (RVOT or basal LV).

Finer definition of tachycardia origin was performed by pace mapping—finding a site at which pacing with the ablation catheter tip could produce a QRS morphology which had 12 out of 12 lead ECG concordance with that of the induced tachycardia itself (Fig 2).

RF energy was then applied at the site defined by the best available pace map, generally employing 40 to 70 volts (15 to 50 watts) delivered from the large tip of the ablation catheter to a topical patch on the patient's back (Fig 3). Energy delivery would be halted immediately if the impedance measured should rise excessively, as this would suggest the formation of a coagulum (charred tissue-clot) which should be scraped off the catheter tip before further energy could be given. Each application of energy would last 30 to 60 seconds.

After each delivery, programmed stimulation was repeated. If tachycardia was not inducible, isoproterenol infusion and PES would be repeated and continued for a total duration of 30 minutes. The procedure was terminated if no single repetitive beat was inducible. All patients received intraveous heparin (3000 units at initiation and 1000 additional units every hour). Post procedure, the patient would be monitored for at least 24 hours. Cardiac enzymes and an echocardiogram would be obtained the next morning to rule out significant myocardial injury or structural damage. Procedural success is defined as noninducibility post ablation. Clinical success is defined as a complete absence of symptoms on follow-up and no arrhythmia documented by ambulatory monitioring.

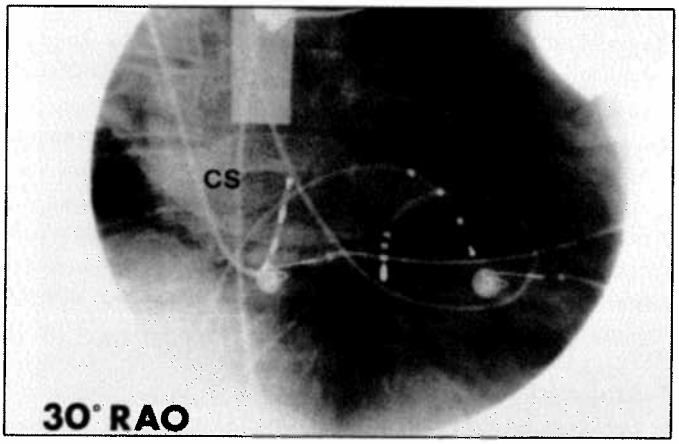


Fig 3.— Catheter positions in Case 1 (in 30 degree right anterior oblique projection). To facilitate a stable catheter position, the ablation catheter was coiled back from the apex. CS=coronary sinus/catheter (outlines the mitral annulus), RF=radiofrequency ablation catheter (with enlarged tip on the basal inferior septum of the left ventricle), RV=right ventricular catheter.

## Results

Five patients underwent the procedure, and their characteristics and procedural successes are listed in Table 1.

Two patients (Cases 1 and 5) had fascicular tachycardia, with the VT origin localized to the LV basal inferior septum. The morphology was right bundle branch block with superior axis (Fig 1). In Case 1, tachycardia was easily inducible by atrial pacing. In the same case, HV interval was -15 milliseconds, consistent with retrograde Hisian depolarization from the left ventricle. In both cases, sustained tachycardia was easily inducible and the induced tachycardia was readily terminable with intraveous verapamil but not with adenosine. Procedural success was very easily attained with 2 RF applications in Case 1 and with only 1 in Case 5. Clinical success continued during follow up.

Three patients (Cases 2, 3, and 4) had RVOT tachycardia, and the VT focus was localized to the RVOT anteriorly, to sites just below the pulmonic valve. The morphology was left bundle branch block with inferior axis (Fig 4). Induction of tachycardia in all 3 cases required isoproterenol facilitation, and the VT induced tended to be nonsustained. Procedural success was quite easily achieved in Cases 2 and 4 and clinical success persisted in follow-up. In Case 3, procedural success was achieved in the first session after 8 applications. A lower-than-average amount of power (15 W) was used because of chest pain associated with each application. Unexpectedly, nonsustained VT recurred the same evening. The patient underwent a second ablative procedure the next day; however, despite vigorous attempts, not a single beat was inducible to serve as a template

**TABLE 1.**—Series of 5 consecutive patients without organic heart disease who underwent radiofrequency catheter ablation for ventricular tachycardia. The cases were arranged in chronological order. Case 1 was ablated on June 3, 1992.

| Case No | Age/Sex | Symptoms | Tachycardia Morphology | Rate | Drugs Tried | No RF (Energy) | Success/ Follow-up |
|---|---|---|---|---|---|---|---|
| 1 | 28/M | p, sob, cp | RBBB, sup | 210 | P,V | 2 (37 W) | + 29 m |
| 2 | 56/M | d | LBBB, inf | 250 | | 7 (50 W) | + 20 m |
| 3 | 28/F | p, cp, d, s | LBBB, inf | 250 | | 14 (15 W) | - 16 m |
| 4 | 55/M | d, f | LBBB, inf | 230 | | 1 (36 W) | + 7 m |
| 5 | 15/M | p, cp, d | RBBB, sup | 170 | | 1 (36 W) | + 7 m |

**Abbreviations**
(cp=chest pain, d=dizziness, F=female, f=fatigue, inf=inferior axis, LBBB=left bundle branch block, M=male, m=months, P=propanolol, Pt=patient, Rate=beats per minute, RBBB=right bundle branch block, RF=radiofrequency applications, s=syncope, sob=shortness of breath, sup=superior axis, V=verapamil).

for repeat ablation. Nonetheless, 6 RF applications were given "blindly" at the same radiological location recalled from the prior study. Afterward, the patient had uniform ventricular ectopy without runs. She was briefly treated with sotalol, then she continued without antiarrhythmic therapy and was essentially symptom-free on follow-up.

In all cases, there was a mild rise in MB CK but the total CK remained in the normal range, consistent with a small amount of myocardial injury. The echocardiograms were all normal, showing no wall motion abnormality, pericardial effusion or valvular abnormality. There were no complications.

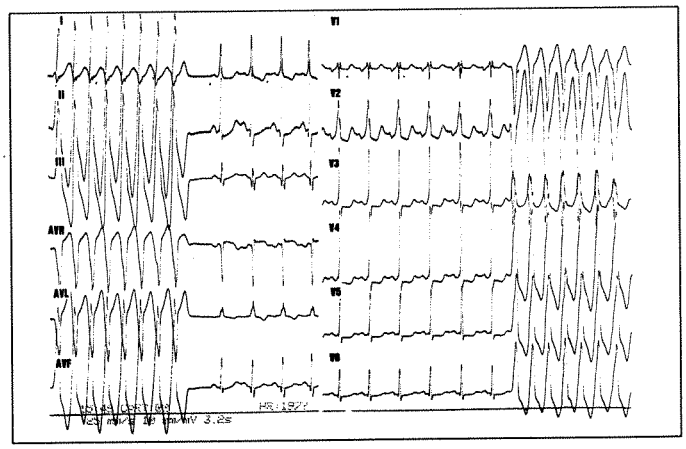


**Fig 4.**—Ventricular tachycardia from Case 2 arising from right ventricular outflow tract. Note left bundle branch block configuration with inferior axis.

## Discussion

**Tachycardia Mechanism and Clinical Features.**—Fascicular tachycardia is rare. First described in 1984 by Belhassen,[11] its location in the left posterior fascicle or left posterior inferior septum is suggested by the morphology of the tachycardia, the range of HV intervals (+15 to –20 milliseconds, consistent with retrograde depolarization of the His bundle from the ventricle) and, as in our cases, by endocardial activation mapping and pace mapping. Typically, such a patient does not have organic heart disease,[12] in clear distinction from the majority of VT patients who have scarred ventricles from prior myocardial infarction or cardiomyopathies to serve as substrate.

Tachycardia mechanism has not been clearly defined. The location in or close to the conduction system may explain why there is such a high prevalence of induction by atrial stimulation (versus most other VTs that are inducible only through the ventricles). This may be possible simply because of geometric proximity. The induction by PES would argue against abnormal automaticity and favor reentry, whereas the very high supressibility by verapamil has suggested a role by afterdepolarizations. Indeed, there has been 1 case of fascicular tachycardia reported with digoxin toxicity in which the tachycardia exhibited certain characteristics that supported triggered activity.[13] The latest evidence favors reentry. This tachycardia has been shown to entrain with constant fusion, and also with progressive fusion at shorter pacing cycle lengths.[14] Addition of verapamil has prolonged the conduction delay during entrainment. Thus reentry with some zone of calcium current dependent slow conduction is a very feasible unifying hypothesis. There is some recent evidence to suggest that the tachycardia arises from the Purkinje network itself.

RVOT tachycardia is probably not as rare as fascicular tachycardia. It was described as early as 1922 by Gallavardin,[15] was named in 1947 by Parkinson and Papp as repetitive paroxysmal tachycardia,[16] and more recently as repetitive monomorphic ventricular tachycardia.[17-19] A typical patient also does not have organic heart disease. This tachycardia generally presents as frequent nonsustained salvos of wide complex tachycardia, frequently with minimal symptoms. In 20% of cases, the tachycardia was sustained (>30 seconds).[20] What distinguishes this tachycardia from the rest is its easy precipitation by stress or exercise. It is not readily provokable by PES and frequently requires isoproterenol for induction, as in our cases. The mechanism is probably abnormal automaticity but triggered activity also has been suggested in view of its response to intraveous adenosine (which antagonizes catecholamine-induced stimulation of cyclic-AMP, believed to have a role in calcium overload-mediated delayed afterdepolarizations and triggering). There is an exceptionally high response to beta-adrenergic blockers, though type 1 antiarrhythmic agents and verapamil also work well should drug therapy be desired.

The prognosis of patients who have these 2 forms of VT is unclear, as both tachycardias are relatively uncommon and there are no long-term follow-up studies. There has been 1 report[21] of a young patient with persistent fascicular tachycardia who

developed a dilated cardiomyopathy and suffered an embolic cerebral transient ischemic attack. The cardiac enlargement reverted following successful treatment with verapamil. Thus, it is probably not benign, though unlikely to be as malignant as the typical VT found in an older patient with prior myocardial infarctions. RVOT tachycardias have a benign prognosis in terms of mortality, with no deaths or worsening of symptoms with follow-up of 6 months to 8 years in 1 series.[18]

**Efficacy of Pace Mapping and RF Catheter Ablation in VT without Organic Heart Disease.**—The lesion caused by an RF application is quite shallow. Its efficacy in fascicular and RVOT tachycardia suggests that the tachycardia focus or the vulnerable portion in the reentrant circuit is at or very close to the endocardium. The zone also is probably quite small, thus allowing for 1 well-placed lesion to annihilate the tachycardia. The lack of scarring in an otherwise normal ventricle would ensure less *distortion* and enable activation mapping or pace mapping to localize the focus more accurately. In our cases, a 12 out of 12 lead concordant pace map virtually guaranteed success (the lone failure in Case 3 might be due to insufficient power employed). By comparison, the circuit in post-infarction or cardiomyopathy-related VT is frequently fairly large and may take various configurations and run very deep subendocardially, thus theoretically more difficult to map and ablate by RF. It stands to reason why ablation for ischemic type VT is far less efficacious, even in the most experienced centers.[22]

**Comparison with other Catheter Interventional Techniques.**—Percutaneous transluminal coronary angioplasty (PTCA) has been widely embraced as a standard of therapy for obstructive coronary artery disease, even though it is, in most cases, a palliative procedure with significant complications including death (.5% to 1%), abrupt vessel closure and infarction (3% to 5%), and other problems such as stroke, renal dysfunction with iodinated contrast load, and vascular damage. This is especially remarkable in the face of a recurrence rate of 25% to 60% at 6 months, with the known fact that there is no difference in prognosis between medical and surgical (likewise PTCA) therapy for 1 or 2-vessel disease—by far the majority of cases undergoing the procedure. RF catheter ablation (for VT and SVT) by comparison is far superior, mainly because it is curative with a much lower complication rate (only 1 myocardial infarction and 1 death reported in the world literature[10,23]) and at a far lower cost (acutely, no surgeon or operating room standby; chronically, no medications, no emergency room visits, no repeat procedures). The more than 90% success rate could be obtained at one-fourth the cost of arrhythmia surgery, with a markedly lower morbidity and virtually no mortality.

**Implications for Diagnosis and Treatment.**—It is quite natural for a physician to assume that a wide complex tachycardia in a young person is supraventricular in origin, especially if there is no evidence of organic heart disease and the tachycardia responds well to intraveous verapamil or adenosine. It behooves the treating physician to be more vigilant for signs suggestive of VT. VA dissociation, if discernable from the 12 lead ECG, is a most valuable clue, whereas other signs such as absence of RS complexes in the precordial leads, QRS concordance, QRS width, etc, while more valuable for the conventional forms of VT[24] are not helpful in diagnosing these 2 specific types. A reasonable index of suspicion should be aroused with any tachycardia having an RBBB/superior axis or an LBBB/inferior axis (especially if the latter should be exercise-induced). Ultimately, electrophysiologic testing may be the only reliable means of elucidation. Precisely defining the mechanism is important because for both of these forms of VT, RF catheter ablation might offer definitive therapy.

## Conclusion

Currently, with cure so readily attainable with the RF ablative technique, empiric drug therapy is no longer the standard of care in most young patients with symptomatic SVT. Our cases demonstrate that there are 2 unusual forms of VT that also are amenable to catheter eradication. When confronted with a wide, complex tachycardia in a young person without organic heart disease, a healthy index of suspicion and a willingness to look more closely may frequently work to the benefit of the patient.

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